Oblique views showed osteophyte encroachment on the left exit foramina of C3-4 and C4-5. No appreciable disc-space narrowing was noted and there was no evidence of cervical ribs.

He was given a course of intensive physiotherapy and fitted with a wrist splint. Six months after myelography, however, there was still pronounced weakness in the left forearm with associated reflex emphasis.

Comment

Side effects of metrizamide when used for both lumbar and cervical myelography have been extensively reviewed.[1-3] Minor side effects such as headache, nausea, and vomiting occur fairly often. Transient hyperreflexia after lumbar myelography with metrizamide has also been reported.[4] One case of areflexia after thoracolumbar myelography was presumed to reflect a direct neurotoxic effect on the cauda equina.[5] Our patient developed acute symptoms shortly after metrizamide myelography. Neurological signs suggested cervical myelopathy, which may have resulted from a direct toxic effect of metrizamide. Spinal canal stenosis, however, may have been contributory.

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Erythema nodosum and infectious mononucleosis

Erythema nodosum is associated with many conditions, the commonest being sarcoidosis.[1] Other examples include drug reactions; inflammatory bowel disease; and some conditions with an established infective cause such as tuberculosis, leprosy, ornithosis, and streptococcal infections. *Yersinia enterocolitica* infection has also been implicated.[2] I report a case in which erythema nodosum occurred in association with infectious mononucleosis.

Case report

A 15-year-old Caucasian girl presented with a six-day history of pharyngitis, arthralgia, lethargy, and a rash on both legs. After the rash appeared she was given ampicillin 250 mg four times daily for four days. There was no history of illness or of drug treatment. The only abnormalities found on examination were an inflamed fauces and erythema nodosum affecting both legs. A throat-swab culture was sterile and the antistreptolysin O titre under 200 IU/ml. Haemoglobin concentration was 12·0 g/dl, erythrocyte sedimentation rate 34 mm in first hour, and white cell count $6{\cdot}8 \times 10^9$/l (6800/mm^3) with 7% atypical mononuclear cells. A Paul-Bunnell test was positive with titres of 224 in saline, 224 with guinea-pig kidney absorption, and under 7 with ox red-cell absorption. Cytomegalovirus titre was under 10, and agglutination tests for *Y enterocolitica* and *Y pseudotuberculosis* were negative. Chest radiography showed no abnormalities, and a Mantoux test gave a grade I-II reaction. There was no response to a Kveim test. Ten weeks later the rash had disappeared and she felt well without needing any specific treatment.

Comment

Infectious mononucleosis has been associated with erythema multiforme[3] but not with erythema nodosum. The present patient showed no evidence of sarcoidosis or any of the other conditions associated with erythema nodosum. The rash occurred during an attack of infectious mononucleosis, a condition thought to be causally related to the Epstein-Barr virus, which is one of the herpesvirus group. A link between herpes-like infection and sarcoidosis was suggested by Hirshaut *et al.*[4] Patients with sarcoidosis were later shown to have the same prevalence of raised antibody titres to Epstein-Barr virus as control subjects but the titres were much higher than those in the controls[5]; patients with non-sarcoid erythema nodosum had about the same antibody titres to all herpesviruses as controls.

I thank Dr W Howel-Evans for permission to report this case, and Dr Bruce White for the serological data.

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Lack of transmission of viral hepatitis type B after oral exposure to HBsAg-positive saliva

Hepatitis B has been transmitted to a human[1] and to gibbons[2] and chimpanzees[3] by subcutaneous or intravenous injections of saliva containing hepatitis B surface antigen (HBsAg). Attempts to transmit hepatitis B by nasal and oral exposure of gibbons to saliva containing HBsAg failed.[2] The possibility that cardiopulmonary resuscitation (CPR) training manikins act as fomites in the transmission of viral hepatitis type B by contact with HBsAg-positive saliva has not been investigated. We report a study in which viral hepatitis type B was not transmitted to people exposed to HBsAg-positive saliva during CPR training.

Subjects, methods, and results

Twenty-two hospital trainees were studied. They had participated in a CPR training programme eight days before one of them developed clinical hepatitis B. The training programme included two all-day classes with intensive practice sessions on five different manikins. All trainees had used a manikin after it had been used by the infected trainee. Ten trainees had also participated in two-rescuer CPR with him during practice or at the time of the final test. Manikin heads had been washed with water and rinsed with 70% isopropyl alcohol after every practice session but they were not dismantled. Their faces and inside mouth area were wiped with a clean absorbent material wetted with 70% isopropyl alcohol after every use. But during two-rescuer CPR there had been no cleaning of the manikin between trainees.

The infected trainee's serum and saliva were positive for HBsAg at the time of his illness. The radioimmunoassay ratios (counts per minute/negative control mean) for the serum and saliva were 40.92 and 7.34 respectively. His saliva was also negative for occult blood. His serum was positive for HBeAg. The trainees were tested for HBsAg and antibody to hepatitis B surface antigen (anti-HBs) two weeks, six weeks, and six months after training. All were negative for HBsAg at two weeks and one was positive for anti-HBs. At six weeks and six months only 17 trainees were tested. All were negative for both HBsAg and anti-HBs and none reported illness. The other four trainees ceased employment with the hospital between two and six weeks after training (including the one with existing anti-HBs). They were contacted at six months and none reported illness during that time. None of the four who ceased employment were among those who participated in two-rescuer CPR with the infected trainee.

Comment

Manikins used in training programmes are a potential vehicle for transmitting hepatitis B. Mouth-to-mouth or mouth-to-nose artificial respiration requires physical contact. Trainees are instructed to disregard sanitary and hygienic precautions to save the victim's life. They therefore commonly practise on manikins contaminated by the oral secretions of others. Manikins may also be contaminated from hands during practice removal of upper airway obstructions. The lack of transmission of hepatitis under these conditions in our study may have

been because HBsAg was not present in the infected trainee's saliva at the time of the CPR class or because the infectivity of the saliva was low. Probably HBsAg was present in his saliva. It usually appears in the blood several weeks before the onset of clinical signs and symptoms. He developed jaundice eight days after the class. Infectivity may be related to the amount of virus present and the mode of transmission. The infectivity of a secretion cannot be determined by the presence of HBsAg, since most of this antigen is associated with subviral particles or defective viral particles.[4] The titre of HBsAg is lower in saliva than in the corresponding serum.[3] Bancroft *et al*[2] found that gibbons could be infected by subcutaneous but not by intranasal or intraoral administration of the same HBsAg-positive saliva inoculum, showing the importance of the mode of transmission.

The cleaning of our manikin heads between use was inadequate to decontaminate for hepatitis B virus. Thus the trainees probably had the maximum exposure that might occur in CPR training. We therefore believe that viral hepatitis type B is unlikely to be transmitted from oral contact with CPR training manikins or other fomites. Mandatory testing of all individuals for HBsAg before CPR training is unwarranted.

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Interaction between disopyramide and practolol

Practolol and disopyramide are commonly used together in treating supraventricular tachycardia, and no adverse interaction has been described. We report two cases in which after practolol and disopyramide were used sequentially to treat supraventricular tachycardia the patients suddenly deteriorated and one died.

Case reports

Case 1—A 56-year-old man was admitted to hospital in March 1979 after 48 hours of chest pain. He had had myocardial infarctions in 1966 and 1974, but had since been well and was receiving no treatment. He was found to have a supraventricular tachycardia of 180 beats/min; carotid sinus massage had no effect. He was given 20 mg practolol intravenously with no response, and then 20 minutes later a slow intravenous injection of 150 mg disopyramide. Within two minutes he developed a sinus bradycardia of 25/min, lost consciousness, and became profoundly hypotensive. He was given 0·6 mg atropine intravenously without an improvement in his heart rate. Later, while a temporary pacemaker was being inserted, his heart rate increased to 60/min. Electrocardiography showed sinus rhythm, right bundle-branch block, left axis deviation, and an acute anteroseptal myocardial infarction. After 24 hours he again developed a supraventricular tachycardia of 180/min; with the pacemaker in position, he was given 150 mg disopyramide intravenously alone, producing a sinus rhythm of 100/min. Thereafter he remained well on treatment with oral disopyramide.

Case 2—A 67-year-old man with widespread vascular disease presented with palpitations, and was found to have a supraventricular tachycardia of 180/min, which did not respond to carotid sinus massage. He was given 10 mg practolol intravenously with no effect and 20 minutes later a slow intravenous injection of disopyramide. After 80 mg had been injected his heart rate reverted to a sinus rhythm of 80/min; this then slowed to 20/min despite intravenous atropine and asystole occurred. He was resuscitated with intracardiac adrenaline, but he remained unconscious and died five hours later.

Comment

In both cases the patients' deterioration was closely related to injection of disopyramide. This drug is now widely used to treat supraventricular tachycardia, apparently with safety,[1] although temporary slowing of the atrial rate by up to 30%, widening of the QRS complex, and transient hypotension have occurred.[2] When given disopyramide without having previously been given practolol, our first patient suffered no ill effects. We therefore suspect an adverse interaction between practolol and disopyramide, and suggest that intravenous disopyramide should not be given after practolol.

[1] Hillis, W S, *et al*, *Journal of International Medical Research*, 1976, **4**, suppl No 1, p 74.
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Vancouver style

All manuscripts submitted to the *BMJ* from now on should conform to the uniform requirements for manuscripts submitted to biomedical journals (known as the Vancouver style).

The *BMJ*, together with many other international biomedical journals, has agreed to accept articles prepared in accordance with the Vancouver style and will be introducing the system from January 1980. The style (described in full in *BMJ*, 24 February, p 532) is intended to standardise requirements for authors and covers text format, presentation of methods and results, use of SI units, and the form of tables and illustrations. All the participating journals have also agreed to introduce a standard form of references.

In future references to papers submitted to the *BMJ* should include: the names of all authors if there are fewer than seven or, if there are more, the first three followed by *et al*; the title of journal articles or book chapters; the titles of journals abbreviated according to the style of *Index Medicus*; and the first and final page numbers of the article or chapter.

Examples of common forms of references are:

[1] International Steering Committee of Medical Editors. Uniform requirements for manuscripts submitted to biomedical journals. *Br Med J* 1979;**1**:532-5.
[2] Soter NA, Wasserman SI, Austen KF. Cold urticaria: release into the circulation of histamine and eosinophil chemotactic factor of anaphylaxis during cold challenge. *N Engl J Med* 1976;**294**:687-90.
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Up to the beginning of October some 100 journals had agreed to accept articles in the Vancouver style, and a full list will be printed early in 1980.